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ANNOUNCEMENT

We are pleased to announce that Dr. Ralph C. Matson, Portland, Oregon, has been elected Chairman of the Editorial Board of the Journal, *Diseases of the Chest* and that he will assume the office of editor-in-chief on August 1, 1941.

Dr. Frank Walton Burge, Philadelphia, Pennsylvania, the retiring chairman and editor-in-chief of the Journal will continue to serve as a member of the Editorial Board of *Diseases of the Chest*.

The other two members of the Editorial Board are Dr. Chas. M. Hendricks, El Paso, Texas, and Dr. Champ H. Holmes, Atlanta, Georgia.

Dr. Burge has asked to be relieved of this office so that he can devote more of his time to his duties as Chairman of the Board of Regents of the American College of Chest Physicians. The responsibilities of this office have increased during the past few years and the growth of the College with its expanding program necessitate Dr. Burge's undivided attention.

Appreciation is expressed to Dr. Burge for his untiring efforts and guidance of the editorial policy of *Diseases of the Chest* during the past four years. His devotion to the end that tuberculosis be eradicated has given added impetus to those engaged in this fight. His zeal and enthusiasm in attacking these problems have brought about many needed changes in our tuberculosis case-finding programs. We are happy to know that his services as a member of the Editorial Board will be available to the new editor-in-chief and his experience will be helpful to those who have now been intrusted with the guidance of the Journal, *Diseases of the Chest*.

EDITORIAL BOARD.

In Review It's not all over yet. There is still much talk and many letters coming across my desk telling of the finest meeting the College has ever had. That was Cleveland this year. It proves the fact that from good preparation comes fine meetings.

To Dr. Joseph C. Placak we extend our kindest thanks for making arrangements for hotel accommodations and meetings and to the members of his subsidiary committees we extend our appreciation. To Dr. Benjamin Goldberg who arranged the Scientific Program and personally conducted the "Information Please" we are also very grateful.

Many remarked that there had never been a finer program on chest diseases, nor one better conducted than this last meeting at Cleveland. The papers will be published in subsequent issues of the Journal on Chest Diseases so that those who were unable to attend may have a full appreciation of these most excellent contributions.

R. C. M.

Tuberculosis is now on Leather An eminent tire manufacturing corporation claims to have placed the farm of today on rubber; the military war lords have mechanized the armed land forces and they now roll over the countryside on wheels; and tuberculosis, to keep apace, and in tune with the forward progress of the times, is now being put more and more, *on leather*. To the pioneers, exponents and workers in collapse therapy, this gradual transformation is due. Many of our tuberculous sick no longer linger and languor away the years in a sanatorium or a mountain fastness; no longer run out their course of a hacking feverish existence at their homes or in an asylum for the incurable. They are, rather, being speedily and at times miraculously restored to exuberant health and taking their place in the world. They once more begin to function in the busy scheme of things of our present day. The arrested, symptom free, re-invigorated individual with his pneumothorax, thoracoplasty, oleothorax or paralytic diaphragm is taking his place in the stream of life as it surges along. They are on the streets of *this* city, *that* city and many of them, on the streets of *many* cities. These may be tourists, salesmen or those engaged in other missions requiring their journeying from here to there. This calls for the necessity of these ambulatory and nomadic patients consulting physicians in distant towns and cities for their refill needs. This in turn demands that these physicians keep abreast of the newer developments in collapse therapy, particularly with an eye to the ease and precision of their technique. Comparisons are said to be odious and they can certainly be in these instances. So pneumothoracists, look to your laurels. *"Tuberculosis is now on Leather."*

C. H. H.

In Memoriam In reviewing the brilliant picture of our accomplishments during the past year, it now becomes our solemn duty to brush this colorful canvas with a stroke of somber hue; to sound unto this pleasing tonal pattern a dark note. We sense, deep within our breasts, the hushed throb of a chant—the litany of death.

Since the last annual meeting, four of our members have departed. They have crossed the bridge and have been enveloped into the misty shadows of the world beyond. The curtain, upon the stage of their fulsome lives, has fallen. It has been said that "by their fruits ye shall know them"—by *their* fruits they were *known*. Upon the honor scroll, history has inscribed these names:

Roland B. Tupper, Fresno, California
Everett E. Watson, Salem, Virginia
Leon Shulman, Los Angeles, California
Jacob Paul Frantz, Clearfield, Pennsylvania

C. H. H.

The Seed is Sown About the first of the year and during the ensuing weeks, there swept over this nation a wave of epidemic influenza. This was the beginning, the planting period of the seeds for a future harvest. The harvest is now being reaped. It is not a harvest of succulent vegetables, of ripening melons; but a harvest of pulmonary diseases. Outstanding among the belated developments or sequelae of this influenza outbreak, is tuberculosis. Carefully investigate the patient who has not fully regained his strength or health from that attack of "flu" or who, since then, has not been able to shake off the cough. Investigate

(Continued to page 235)

Address*

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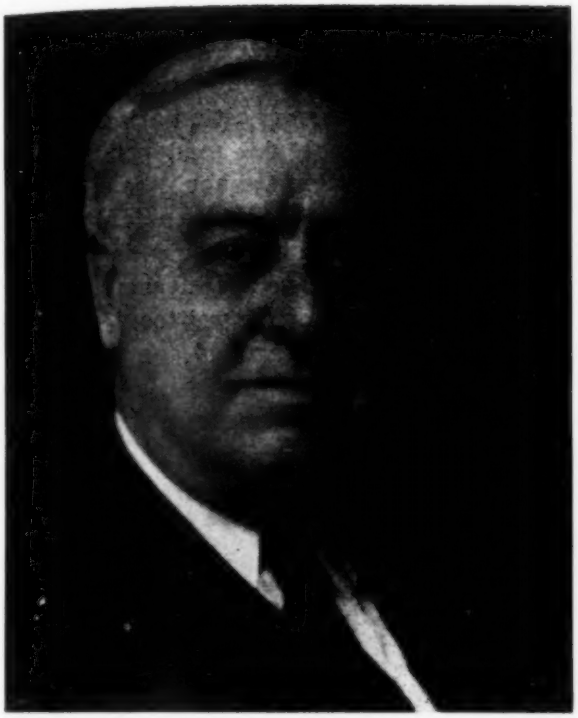

Fellows of the College, ladies and gentlemen, we are all familiar with the homely old saying, if only our foresight was as good as our hindsight. Unfortunately, most of us must learn by experience, but to a few souls is given an ability beyond the average, to see ahead, souls like that of the wise man in the East whose constant prayer was that he might see today with the eyes of tomorrow. Surely, some such prayer was in the hearts of those who first visioned the American College of Chest Physicians as a power for good, and those who since have endeavored and are endeavoring, to maintain that tradition. Such men are interested not in what the College can do for them personally, but in what the College can do for others. But a strong foundation is not enough. Only by using bricks, strong with generosity, thoughtfulness, wisdom and foresight, can we build an edifice that will stand, and be worthy of the foundation already laid.

There is much to be done, no matter how much has been done; there is still so much to be done that we must not spend our time dwelling on the past, but rather, like the runner in a race, focus our attention on what lies ahead. Nevertheless, being all fashioned of a somewhat weak dust, it is easier to go forward when we hear the populace cheering behind. And with this in mind it is fitting that I relate, more briefly I am afraid, than true deserts call for, some of the accomplishments of the last year, chiefly through the committees which were appointed to help guide the destinies of the College.

This is the seventh annual convention of the College. In this relatively short period of time we have achieved a degree of success which should be most gratifying to its founders. We are now on a stable basis financially and professionally. The reports from the various committee chairmen will indicate satisfactory progress along our established lines of endeavor. No greater stimulous to realization of ideals of purpose is needed than the inspiration of a group of physicians who will carry the task on to ultimate completion. Such is the present status of the College.

Some of the downright optimistic fellows last year envisioned a membership of 1,000 fellows and associates by June 1941. That meant a gain of nearly 100 per cent, which seemed impossible of fulfillment. Our qualifications are high, and our field is limited. You will learn from others that this magnificent goal has to all intents and purposes been reached. We are justly proud of this record, and prouder still that the names of practically all the outstanding chest physicians of the nation are on our rolls. Our potential ability to carry out the high aims and objects of the College is thus beyond doubt. I have no doubts regarding future success, with every fellow giving his utmost of effort, ability and encouragement. There are still eligible candidates outside the fold, and the drive should not lessen until they are safely within the College.

The organization of Chapters of the College, in Illinois, New York, New Jersey, Missouri, Ohio, Pennsylvania, Texas, the Northwest, Cuba, and in formation in several other

* Seventh Annual Meeting, American College of Chest Physicians, Cleveland, May 31-June 2, 1941.

states, represents most worthy progress. Close affiliation of the Chapters with the various state medical societies gives us and our work added impetus and strength. Some plan must be devised for giving the benefits of chapter organization to states with less than the minimum number of Fellows required to form Chapters.

Financial support from the College treasury to these state or district Chapters may be requested. The general opinion seems to favor the preferable procedure that these groups undertake to finance themselves as independent affiliates.

The College is accumulating a supply of decrepit old gentlemen known as Past Presidents. They are a distinct asset, but may become a liability unless kept at work. Your action last year in making them members of the Board of Regents without portfolio was most commendable. I trust that there may be found a place in the College where full utilization of their talents may be exercised in careful advisory capacity. I doubt that they should be appointed to important positions, as many young, active men are coming on who must receive the necessary training to advance the work of the College.

The plan of organization of the American Medical Association has stood the test of time, hence in my opinion we should imitate some of their procedures. I refer to the establishment of Councils instead of standing committees. Appointments for a term of years will insure continuity of service and effort which always assures progressive action. As at present constituted, it is possible that the personnel of all committees may be entirely changed from year to year.

One might suggest a Council on Sanatoria; a Council on Tuberculosis Education; a Council on Scientific Programs, or Scientific Assembly; a Council on Advancement of Tuberculosis Organization.

If so constituted, we would need then relatively few committees. All of us must have a Nominating Committee, and very few others.

Our rapidly expanding numerical strength indicates greater financial security. The report of the Treasurer regarding our finances which you will soon hear, will be most gratifying. Removal of our headquarters to Chicago has proved to be distinctly advantageous from many viewpoints, although the cost of operating such an office has necessarily increased the budgetary expense.

Closely allied with this expansion is the question of establishment of a scientific journal as the official publication of the College. Immediately the question arises whether the present journal, designed primarily for the general practitioner, should be continued. To determine this will require a careful study of relative values which should doubtless be a proposition for thorough investigation and study by a competent committee.

Less and less is heard of competitive organizations in our field. Really, there can be no competition in our efforts to control tuberculosis; there is plenty for all to do. Our position of leadership is becoming increasingly apparent but we must be on our toes to live up to our obligations. The fellows who advised that we go ahead with our own program to the best of our ability were wise counsellors indeed. Their judgment is fully vindicated and our position is stronger and more secure than ever before.

Such, briefly, is a resume of the year's record of tangible accomplishments of the American College of Chest Physicians. Of the intangible accomplishments which have emanated from your endeavors, neither you nor I have the power to speak. If we have made progress, there will be cheers, whether we hear them or not. If we have made failures, they will be forgotten. Who remembers, when the runner in the race crosses the goal line, that he once stumbled and fell?

My year as President of the American College of Chest Physicians has meant more to me than I can put into words. I stood before you a year ago, humble and a bit frightened. I stand before you today, still humble, but no longer frightened, only grateful for the wholehearted support you have given me, and for what you have done for the College. I am a bit awed at the thought of what we can accomplish individually and collectively in the years to come, if we all pray constantly that we see today with the eyes of tomorrow.

Pulmonary Actinomycosis Complicated by Pneumothorax Treatment

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Introduction

Pulmonary actinomycosis in human beings is still a comparative rarety in the United States. On the European Continent, and particularly in Germany, Austria, and Russia, lung infection with actinomycosis is seen fairly often.

The old theory that actinomycosis is contracted by chewing straws and grasses is at present held not to be well founded; at least from the clinical standpoint, this hypothesis in cases reported in the literature has not been substantiated. Aspiration as a means of infection was definitely proved in a number of cases by finding aspirated foreign bodies such as pieces of carious teeth surrounded by the organism. The infection apparently takes place by direct aspiration of infective material through the air passages and occasionally by way of metastasis through the blood stream. The cavity of a carious tooth appears to be a frequent seat of actinomycotic infection. It is uncertain whether or not the actinomyces are able to pass through an intact mucous membrane. However, a number of cases are on record in which infection followed wounds or injuries. During the World War, for instance, we saw a number of cases, where soldiers who had sustained abdominal and pelvic injuries were later found to have actinomycosis, probably as a result of having been bedded on grain stalks and straw. The actinomyces, in these cases, undoubtedly entered by the open wounds.

Diagnosis

The clinical diagnosis of pulmonary actinomycosis is not an easy one to make. The symptomatology of the disease does not have any specific characteristics. It may resemble that of pulmonary tuberculosis, or in some cases, the clinical manifestations may suggest malignancy or syphilitic infection of the lungs. The disease apparently begins as a chronic bronchitis or pneumonia with evidence of a spreading, caseating process in the lungs and later shows a definite tendency to produce large solitary pulmonary abscesses or cavities surrounded by rather dense caseous infiltration and exudation. Not until metastatic sinuses (abscesses) develop in different parts of the body can the true nature of the disease be suspected and recognized. Even then, repeated histologic examinations, cultures, and exploratory excisions are required in order to arrive at a correct diagnosis. Roughly, there apparently are two main clinical stages in the development of pulmonary actinomycosis. The first stage of the disease is characterized by the development of a bronchial catarrh with a moderate amount of expectoration. During the second stage of infection, the process is extended to the lung tissue, productive cough increases in severity and annoyance, sputum becomes suppurative, and is expectorated in large quantities. At this period, if the disease remains unchecked, the limits of the lung tissue are passed and the infection attacks the pleura. A moderately strong fetid odor may become manifest at this stage of the disease, but this symptom in itself should not be considered pathognomonic of pulmonary actinomycosis and no specific value should be given to that symptom in regard to the diagnosis. Any severe wasting disease may produce that particular symptom. Only when actinomyces are found in the sputum, in the pleural exudate, or in the pus from the sinuses, may the true diagnosis be suspected. Roentgenoscopy more often misleads than helps. It often leads to incorrect diagnosis because the roentgen picture will strikingly resemble pulmonary tuberculosis, bronchiectasis, malignant tumors, or pulmonary abscesses, and the differentiation is extremely difficult. In other words, correct diagnosis is based mainly on the bacteriologic demonstration of actinomycotic filaments on ordinary smears and repeated cultures. The appearance of the characteristic yellow grains, so-called sulphogranules is not a necessary prerequisite for a clinical diagnosis since the

infection may be caused by a type of fungi devoid of that characteristic. The patient's history may be of tremendous help. Extraction of infected teeth prior to developing the infection should always be considered as a possible clue to the diagnosis. The frequency with which actinomycosis follows dental procedures should be emphasized. The disease may develop following dental extraction, treatment of pyorrhea, and various other inflammatory conditions of the mouth. It is important, therefore, to remember that all types of dental infections may be associated with actinomycotic infection. However, it is no less significant to keep in mind that ray fungus is known to be a frequent inhabitant in the mouth of many individuals with good oral hygiene.

Prognosis

From the study of cases of pulmonary actinomycosis reported in the literature one may easily conclude that the prognosis is, as a rule, unfavorable. Almost all authorities agree that when the disease has metastasized to different parts of the body, the prognosis is always poor. Practically all cases of extensive pulmonary actinomycosis in the lungs which were reported in the English and American literature had fatal results. Apparently, in the pulmonary form the prognosis is less favorable than in other forms because, up to the present time, no effective therapy has been offered. Iodides, thymol and roentgen therapy were reported helpful, but seldom were effective in regard to cure. Sulfanilamide was recently suggested, but no cases have been reported where sulfanilamide therapy were used.

In our case, sulfanilamide produced good results for a limited period of time, but the treatment was not sufficient to accomplish a cure. The therapy, therefore, remains a difficult problem. The difficulty lies in the fact that thoracic cases are usually diagnosed when there is already extensive involvement of the lungs and at that stage even heroic measures are of little help. Early recognition of the disease is, therefore, of tremendous importance as far as prognosis is concerned.

Case Report

H. M., white, male, age 49, was admitted to Glenn Dale Sanatorium on November 29, 1939. On admission he stated that his health had failed generally since the middle of July, 1939, when on the advice of his dentist all of his teeth were extracted (17 in number) under novacaine anesthesia. Apparently there was an extensive pyorrheal infection; x-ray of teeth taken previous to extraction showed evidence of destruction of alveolar processes and diseased periapical regions in a number of them. He also had a severe cold, which, according to his statement, lasted for over three weeks. By September, 1939, he commenced to have pain in the left chest and about the same time developed a persistent productive cough. He felt quite tired, had numerous night sweats, lost considerable weight. He was put to bed and as he did not show any improvement, he entered a general hospital in November, 1939. A diagnosis of pulmonary tuberculosis with cavitation in the left upper lobe was made and left artificial pneumothorax instituted. However, the record of that hospital shows that no acid-fast bacilli were found (several concentrated specimens) and that "spores and mycelia present in sufficient number to suggest the desirability of a culture being made." Shortly after institution of pneumothorax he was referred to Glenn Dale Sanatorium. A total of five refills were given before he was admitted. At the time of admission to Glenn Dale he had a severe productive cough; temperature was elevated, ranging from 98° to 103°; pulse rate from 88 to 132; respirations from 24 to 36.

Physical Examination

The patient's condition on admission was very poor. He appeared older than the given age, showed extreme emaciation with a tremendous loss of subcutaneous fat and wasting of the muscles. He was surprisingly alert, fully oriented, and quite cooperative. On the lower left posterior part of the skull there was an infected area with some purulent discharge. Shortly after admission smaller infected areas were noticed over the prominence of the first joint of the left thumb, right lower leg, and left lower chest posteriorly. All of these cutaneous lesions were red, somewhat raised, punched out ulcerated nodules, exudating purulent material. The skin margin surrounding the ulcerated areas was indurated and showed a reddish purple discoloration. The cervical and epitrochleal

nodes were not palpable. All the teeth had been removed. The gums showed no evidence of any lesion.

Lungs—The body thorax was rather long and narrow. The left side did not perceptably expand, though some motion was felt on palpation. The fremitus was intense over the right side but poorly felt over the left, front, and lateral portion. On percussion the note was tympanitic over the entire left chest, front and lateral, and almost equally so over the right chest down to the costal margin, the liver dullness being obliterated. On auscultation, breath sounds on the left side were heard faintly over the upper, inner chest, and scattered over the entire left chest occasional rather tinkling rales were heard. Over the right chest anteriorly the breath sounds were markedly accentuated down almost to the costal margin. Whispered and spoken voice sounds were suppressed over the left chest but increased over the right chest anteriorly. The posterior portion of the chest showed signs about the same as in front.

Heart—The apex beat was not localized. The sounds were somewhat distant, rapid, regular, without any definite murmurs or arythmias. The cardiac dullness could not be delineated; the pulses were equal and regular.

Abdomen—The abdomen was scaphoid in shape without any adipoise tissue. No tenderness or masses. The liver, spleen, and kidneys were not felt.

Several weeks following admission there appeared over the insertion of the left deltoid a rounded, soft, fluctuant, slightly purplish mass which was a bit tender; also a diffused swelling about the right elbow joint, where motion was limited, with some tenderness. The capsule of the joint seemed definitely distended. Similarly, the left knee joint was markedly distended, extremely tender, and painful with definite limitation of flexion. The patella was high and fixed.

Laboratory Findings

Sputum produced in large amount was thick, tenacious, muco-purulent in character. It was persistently negative for tubercle-bacilli on direct smears, concentration, and culture. The stained smears revealed numerous long, granular, interlacing, branching filaments with no ray formation. Macroscopically no granules could be detected. In the earlier specimens no club formation was noted, but as the disease progressed, club-shaped swellings were found on the ends of the filaments. The filaments were gram-positive, retaining the stain unevenly. When stained by Ziehl-Neelsen method, the granules of the filaments retained the acid-fast stain while the main body did not. When the sputum was inoculated on blood agar plates and Hohn's egg media and incubated for 36 hours at 37° C, a white, dry, chalky, tough, somewhat raised growth was obtained on the surface. After several days there was a pronounced wrinkling of the growth and the color deepened to a light orange.

Attempts to grow the organism under anerobic conditions were unsuccessful. This organism with the exception of being non-acid-fast had all the characteristics of that isolated and discussed by Eppinger in 1891[1] and now generally classified as actinomyces asteroides. Aspirated material from the abscesses on the arm, knee, scalp, and other infected areas showed identical characteristics. They did not reveal the presence of any granules. All smears showed the actinomyces filaments and pure cultures of actinomyces asteroides were obtained repeatedly. No tubercle bacilli nor any other organisms except actinomyces asteroides were obtained at any time on smears or cultures (blood agar, Hohn's media, broth). Apparently the strain of actinomyces obtained from sputum and purulent material aspirated from several metastasized abscesses were identical from bacteriological and cultural standpoints, that is, they showed definite characteristics of actinomyces asteroides except that they took the acid-fast stain very poorly. Guinea pig inoculated in the peritoneum with 3 cc. of the concentration of the purulent material and killed six weeks later showed no evidence of tuberculosis or actinomycosis.

Blood—Due to the fact that the patient was on a prolonged sulfanilamide regime, practically daily hemograms were taken. They ranged as follows:

Hg:	40-63%
R.B.C.	2,460,000 — 3,890,000
W.B.C.	11,600 — 44,000

Differential

Leukocytes:	8—76%
Monocytes:	1—14%

Young forms:	2— 9%
Band forms:	5—39%
Segmented forms:	5—74%
Neutrophiles	17—82%

Occasional eosinophile and basophile.

Blood chemistry

12/22/39—Free blood sulfanilamide 10.6 mgs.
12/26/39—Free blood sulfanilamide 10.1 mgs.
1/12/40—Free blood sulfanilamide 13. mgs.
Leukocytic Index: 100 plus.
Sedimentation rate ranged from 24 to 29.5 mm. per 60 minutes.
Wassermann negative.

Urine—Amber in color, always cloudy in appearance, negative for sugar, positive for albumen in each specimen. Microscopically, a few leukocytes and occasionally some fine granular and hyalin casts were seen.

P.S.P. test—A total of 93 per cent of dye excreted in 2 hours following injection of dye (shortly after admission).

Roentgenography—The film taken on admission showed dense infiltration of the upper half of the right lung. On the left side the film showed small pneumothorax collapse of the lower two-thirds of the lung with a horizontal fluid line, approximately reaching the upper border of the fourth rib anteriorly. The upper left lung area was filled with rather soft infiltration. Several annular rings suggesting cavities were seen in that area. There was no evidence of bone involvement on admission. Several weeks later, however, the left knee became red, painful, markedly swollen; osteomyelitis rapidly developed with subsequent complete destruction of the knee joint.

Course in the Hospital

The patient was admitted in critical condition. He had an artificial pneumothorax instituted before admission and apparently developed fluid in his chest. The collapse therapy was done on the supposition that the lesion was tuberculous. As soon as the diagnosis of actinomycosis was proved, intensive iodine treatment was prescribed. As no appreciable results were obtained in a period of ten days, the patient was put on sulfanilamide treatment supplemented with nicotinic acid and Vitamin B-1. Transfusions were frequently given. During the first six weeks of this treatment the patient showed considerable improvement. His temperature came down to normal; pulse and respirations were normal; he gained weight and in general was very comfortable. The abscesses were healing rather rapidly and the little discharge which was still present did not show any more actinomyces on ordinary smears and cultures. The results of treatment up to this time were extremely encouraging. He coughed very little and the amount of expectoration decreased greatly. X-ray also showed evidence of considerable clearance on the right side, but the left side showed a gradual increase in the amount of collapse of the lung. Apparently, there was a tension pneumothorax with a definite simultaneous increase in the amount of fluid in the pleural cavity. The fluid proved to be yellow, purulent material with a putrid odor, containing many gram-positive cocci. No tubercle bacilli or actinomyces were found in the fluid.

The air and fluid were aspirated several times in order to expand the lung. We were not successful in obtaining that result. His left pneumothorax continued to increase; also the fluid in the pleural cavity. In the latter part of January his temperature began to elevate to 101°, 102°. Several additional metastatic abscesses developed from which we again could obtain actinomyces asteroides on smears and cultures. At that time the treatment had no effect whatever. He finally developed a generalized edema, severe dyspnea, and a rapid down-hill course followed. He died on March 22, 1940.

Autopsy Report

The patient was a white, markedly emaciated male, whose estimated body length was 5 ft. 6 in., and weight approximately 120 pounds. On the head there was a purplish area in the left occipital region, 5 cm. in diameter, which contained 3 open wounds, the largest being 3 mm. in diameter. The mucous membrane was markedly pale. There was grade 4 pitting oedema of the right hand, and grade 1 pitting oedema of the left hand.

External marks—An area a few mm. in diameter which exuded pus, foul smelling, and of brownish nature over middle one-third of the left femur. There were several puncture wounds in left axilla. The left knee was markedly swollen. Medially and laterally there were 2 incisions 1½ cm. long from which

exuded a light brown, puriform material which was foul smelling.

Peritoneal cavity—There was about 35 cc. of clear straw colored fluid present. There were no adhesions. The lymph glands were slightly enlarged. The peritoneum was smooth and shiny.

Pericardial cavity—There was about 20 cc. of clear straw colored fluid present.

Pleural cavities—In the left pleural cavity there was approximately 750 cc. of dark green, thick, purulent fluid. The right pleural cavity contained no fluid; there were numerous fibrous adhesions present at the apex.

Heart—The heart was normal in size. The pericardium was smooth, and all valves were normal. The heart muscle was firm and of a brownish color. The coronaries were tortuous, and showed grade 1 atheroma.

Lungs—The entire left lung was bound down and atelectatic. The pleura was thickened to about 3 mm. and on section the lung seemed to have numerous, scattered, yellowish-gray areas, mainly arranged about the terminal bronchioles. There were numerous bronchiectatic areas throughout the entire lung substance. The upper one-third consisted of a hollowed-out, trabeculated, roughened cavity which was empty. Several of these yellowish areas were broken down and exuded a yellowish-brown material.

The right lung was markedly voluminous, and crepitant throughout. On section the upper lobe revealed a single isolated lesion approximately 5 cm. in diameter which on cutting into revealed several small communicating cavities, which were lined by a smooth membrane and contained purulent material. The middle lobe was atelectatic and almost completely airless. The lower lobe was a purplish red in color, quite firm, and in its lower half and on section considerable sero-sanguinous fluid could be expressed.

Spleen—This was enlarged to 1½ times the normal size. The capsule was smooth, and on section showed considerable congestion and several calcified pin-head sized yellow nodules.

Liver—Normal in size, considerably congested. On section the liver was chocolate brown in color and showed grade I nutmegging.

Adrenals—Normal.

Kidneys—The left kidney was enlarged 1½ times, and the capsule stripped with ease. On section, the organ was intensely congested. In the pelvis and calyces were found some yellowish-brown, finely granular, hard substances. The right kidney, normal in size, showed similar findings.

Bladder—Contained stones similar to those in the kidneys. Otherwise, normal.

Prostate—Enlarged and contained a few adenomatous nodules.

Pancreas—Normal.

G. I. Tract—Normal.

Knee—On opening into the left knee joint, there was crumbling of lower end of left femur and considerable erosion was present. The joint cavity contained about 300 cc. of green purulent material.

Microscopic Examination

Heart—The fibers were vacuolated and torn asunder by freezing.

Aorta—Showed atheromatous placques.

Lungs—(14 sections). All but two showed the same picture—discrete and confluent granulomatous lesions characterized by extensive proliferation of fibroblasts causing extensive scarring, obliteration of bronchioles and alveoli, and carnification. The granulation tissue was infiltrated with polys and macrophages in some areas, but not in all. A few sections showed frank abscesses walled off by granulation tissue. About the periphery of these abscesses and in other areas were peculiar deeply eosin staining hyaline necrotic areas about 1/10 mm. in diameter surrounded by invaded "epitheloid cells" but no giant cells. Everywhere associated with the granulomatous lesions appeared a very large characteristic and striking circular to oval cell-like or yeast-like bodies 4 to 6 times as large as a macrophage. This body had a very dark staining "blotch-like" nucleus quite unlike that of a tissue cell (no chromatin granules) which was often surrounded by a narrow clear zone. The cytoplasm was abundant in amount and was either dark and indistinct or was distinctly granular. In some cells the granules were very distinct. There was no definite ectoplasm. No ray fungi or sulphur granules were seen anywhere. No tuberculosis was present. One section showed a diffuse polymorphonuclear infiltration — the typical picture of terminal bronchopneumonia.

Spleen—Large amount of intracellular hemosiderin—hemosiderosis.

Kidney—Architecturally normal.

Prostate—Well marked hyperplasia associated with dilated acini lined by very flat epithelium. Pus was seen in many acini.

Anatomical Diagnosis

Left pleural emphyema.

Fungus infection—left lung and right upper lobe with cavitation and bronchiectasis.

Osteomyelitis—left femur with acute septic arthritis—left knee.

Sinus of scalp.

Renal and bladder calculi (acetyl sulfapyridine?).

Hepatic and renal congestion.

Clinical Diagnosis

Generalized Actinomycosis complicated by left pneumothorax.

Comment

A case of advanced bilateral pulmonary actinomycosis complicated by artificial pneumothorax with a number of metastases to different parts of the body is reported. In reviewing the literature, one finds very few cases of actinomycosis in which the disease is limited to the lung bed. It is, therefore, of almost importance to give to the clinician, and particularly the one who is primarily interested in respiratory diseases, a syndrome on which the diagnosis of this condition may be made. History of teeth extraction, indefinite persistent pain in the chest, associated with a productive cough of a mucoid or purulent sputum, loss of weight, weakness and emaciation, all of these symptoms should be considered of sufficient importance to rule out actinomycosis, particularly so if the sputum remains persistently negative for acid-fast bacilli. There is usually secondary anemia, moderate leukocytosis, and a considerable increase in granulocytes. Temperature is septic with an increasingly associated rise

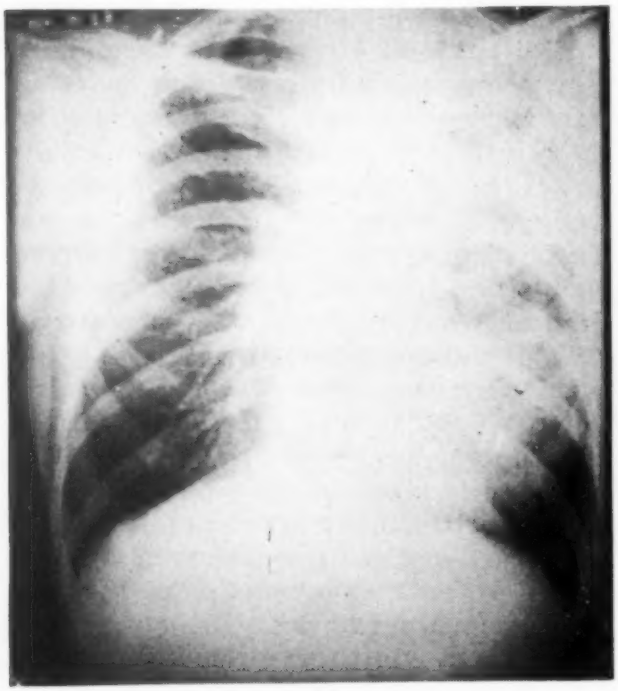

FIGURE 1

X-ray taken before any treatment was instituted. The right lung field appears to be free from definite pathology except for peribronchial infiltration in and around the hilum. The upper three-fourths of the left lung is filled with a dense, nearly completely opaque, caseofibrous infiltration. There is a lighter area in the upper fourth suggesting a large cavity. The pathology of this film strikingly resembles pulmonary tuberculosis.

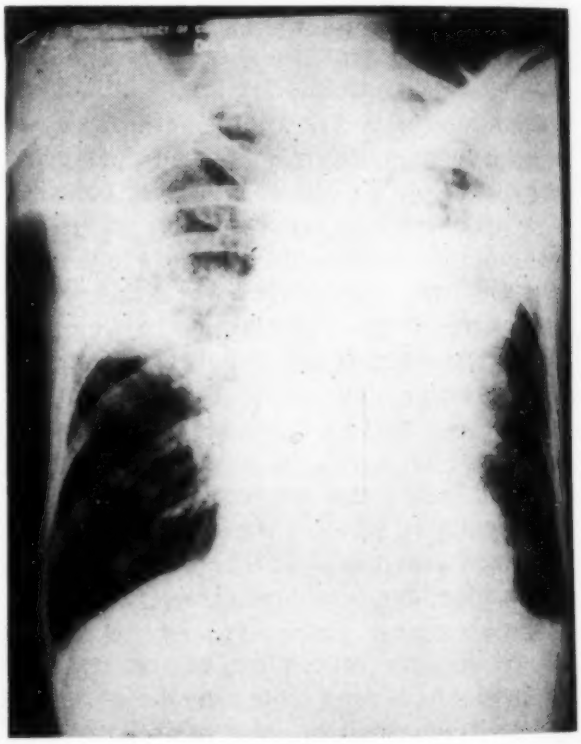

FIGURE 2

This plate was taken following institution of left pneumothorax on the assumption that the pathology was pulmonary tuberculosis, advanced lesion with cavitation. The left lung field shows a 20 per cent marginal pneumothorax collapse below the third rib with a small amount of fluid in the costo-phrenic angle. The collapsed lung is nearly completely opaque except for the upper third where several areas of lesser density, suggesting cavities, were seen. Following institution of pneumothorax there apparently appeared a spread of pathology to the right and the plate shows the upper half of the right lung filled with a dense caseo-fibrous infiltration.

in the pulse rate. The symptoms of actinomycosis of the lung may resemble that of bronchitis, bronchopneumonia, or pulmonary abscess. However, pulmonary tuberculosis is naturally the condition with which it is most apt to be confused because the clinical picture of pulmonary actinomycosis and tuberculosis simulate each other very closely. The differential diagnosis will depend entirely on the laboratory findings, the recovery of specific fungus on ordinary smears and cultures. Primary actinomycosis of the lung is usually a bronchopneumonic form. Cough and expectoration are practically identical with those of a tuberculous lesion. The roentgenogram is also strikingly similar to that of pulmonary tuberculosis of advanced stage.

Pathologically, actinomyces produce in the human organism such disintegration and destruction of normal tissue that the process has almost malignant characteristics. When actinomycosis affects the lungs, primarily, it tends to produce large solitary abscesses (cavities); the infection is also characteristically associated with multiple metastatic sinuses.

Early recognition of the disease is of tremendous importance. Adequate sulfanilamide treatment, supplemented by small transfusions, in that stage may effect recovery.

Pneumothorax therapy is not indicated and should not be instituted in cases where diagnosis of actinomycosis is suspected.

Glenn Dale Sanatorium.

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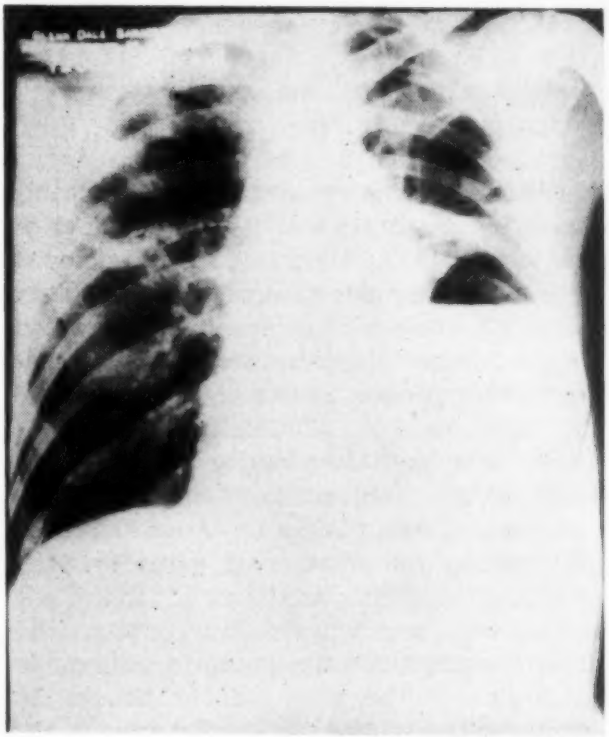

FIGURE 3

This plate was taken following vigorous sulfanilamide treatment. The right lung field has cleared considerably, leaving some evidence of moderate infiltration in the upper two-thirds. The left side shows increased tension pneumothorax limited below at the level of the third anterior rib by a horizontal fluid line. The collapsed lung is adherent to the apex at the level of the second interspace. The infiltration in this area has become much less dense.

We wish to thank Dr. C. W. Emmons, Senior Mycologist, U. S. Public Health Service, for his interest and valuable cooperation in helping to identify the cultural characteristics of the isolated organism. We also extend our appreciation to Mr. Tomas Jefferis, Senior Medical Technologist of the Glenn Dale Sanatorium, whose untiring efforts and assistance were most helpful in establishing the correct diagnosis.

THE SEED IS SOWN—*(Cont. from page 226)*

them by x-ray examination and repeated examination of the sputum. Viewed from the narrowed vision of epidemiology; an attack of influenza is not strictly the etiological factor in tuberculosis; but clinically it frequently may be the starting point at which the spark of a latent or dormant infection is fanned into burning flame. It frequently serves to orient the patient as the time of his departure from normal health. These individuals are now purchasing tonics, vitamines or coming to your office for a helpful suggestion. Keep tuberculosis in mind and look for it. You will find it in a surprising number of instances. The harvest is coming in. Reap it well!

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The Reflex and Chemical Control of Respiration*

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Of those systems whose function is accomplished by rhythmic activity, the control of none is of more interest to both physiologist and clinician than is that of the respiratory system. This is especially true in a day when so much of clinical procedure concerns measures such as general anesthesia, oxygen therapy, post-operative administration of CO_2-O_2 mixtures and attention to the problems presented by the anoxemia of cardiac failure.

The act of respiration is designed to accomplish an interchange of gases between alveolar air and blood to the extent that blood leaving the lung will contain oxygen and carbon dioxide under tensions and in amounts best suited to the needs of the tissues. In the normal individual the interchange is accomplished in the presence of surprisingly constant partial pressures of O_2 and CO_2 in the lungs. Depth and rate of respiration are so adjusted that tidal air is enough greater than the volume of the dead space to permit just the right amount of alveolar ventilation to maintain an optimal composition of alveolar air. Interchange of gases across the alveolar walls occurs entirely by the process of diffusion, a fact which makes it unnecessary for filtration of tissue fluid to occur and which explains the lack of a need for pulmonary capillary pressure ever to exceed the osmotic pressure of the plasma proteins.

The regulation of respiration is accomplished by the rhythmic discharge of impulses from specific groups of neurones of the reticular formation of the medulla oblongata. Early work resulted in the belief that the respiratory neurones were grouped closely into a respiratory center. The experiments of Pitts, Magoun and Ranson[9] have shown that there are two groups of respiratory neurones on each side of the medulla and that stimulation of the more ventral and caudal of these results in maximal inspiration, while stimulation of the other group results in maximal expiration. The former area may be stimulated continuously to the extent that inspiration is maintained until death of the animal occurs, while in the absence of the latter, respiration becomes gasping. Thus the respiratory "center" as it is still called, consists of neurones of the reticular formation lying in the ventral portion of the medulla, some of which are concerned with innervation of the muscles of inspiration, others with innervation of the muscles of expiration and still others with innervation of accessory muscles of respiration.

Of fundamental interest is the origin of the impulses in the respiratory neurones of the medulla. Although the normal respiratory rhythm is known to depend largely on incoming afferent impulses, it is important to know whether the neurones are capable of maintaining an intrinsic rhythm independent of impulses from other sources. Evidence seems to favor existence of such an intrinsic rhythm after experimental removal of all afferent nerves, as well as fibers from higher levels. The complete functional isolation of the respiratory neurones from all possible sources of incoming impulses is obviously difficult, and to that extent it is difficult to be certain that these cells possess an intrinsic rhythm. Although no conclusive statement can be made as to its cause, we know that in order for the rhythm to occur, oxygen must be supplied to the cells, and that any chemical change in the blood associated with a rise or fall in hydrogen-ion concentration results in a corresponding variation in the frequency of discharge of impulses.

The individuality of inspiratory and expiratory groups of neurones of the reticular formation may be correlated with the observation that in oxygen-lack, expiratory movements are first to disappear. This suggests that the expiratory cells have a lower threshold to the depressing influence of anoxia than do the inspiratory cells. On the other hand, the inspiratory cells have a lower threshold to the stimulating effects of carbon dioxide as shown by the effects of breathing

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gradually increasing concentrations of this gas, the first noticeable change being an increase in the inspiratory phase of respiration.

It is evident then that the respiratory neurones of the medulla oblongata may be divided into inspiratory and expiratory groups, that evidence favors an intrinsic rhythm of these cells, that their normal activity is dependent upon oxygen and that increase in their H-ion concentration is capable of augmenting the activity.

The Reflex Control of Respiration

In such conditions as edema of the lung, it may become impossible to establish normal interchange between alveolar air and blood to the extent of maintaining normal O_2 and CO_2 content of arterial blood. This result is based largely on the development of greater diffusion distances incident to the edema of the alveolar wall. It presents a need for greater gas pressure gradients across this wall, i.e., increased alveolar O_2 and reduced alveolar CO_2. These changes in alveolar gas partial pressures may be accomplished of course by increased ventilation. The type of breathing found in congestive cardiac failure with pulmonary edema is, however, of a rapid shallow type in which tidal air may actually be reduced below the volume of the dead space. The result is inadequate ventilation of the alveoli, reduction of their oxygen content, anoxemia, and anoxia of the respiratory neurones with consequent tendency in the direction of depression. The anoxia, of course, affects not only respiratory neurones but also the vasomotor, cardiac and other centers to the extent of promoting respiratory and circulatory failure, results which are readily explained on the basis of anoxia. The reflexes responsible for the shallow respiration of lung edema as well as of other conditions such as emphysema are then of great concern to the physician.

The role of reflexes in the control of respiration is not made less important by recognition of an intrinsic rhythm of the respiratory cells. In fact, the respiratory rhythm observed in the normal subject is largely dependent on impulses arriving over specific afferent fibers. To illustrate this dependence we need merely section both vagus nerves and observe the prolonged inspiratory phase and reduced rate of respiration. Similarly, it has been reported that in anesthetized animals, section of the 9th nerve branches to the carotid sinus and body frequently results in cessation of respiration. Thus even though respiratory cells may be capable of an intrinsic rhythm, it is apparent that the respiratory pattern normally obseved depends largely on reflexes. As we shall see, the adjustment of respiration to meet the needs of the tissues also may depend upon reflex as well as central factors.

The reflexes specifically concerned with control of respiration may be considered conveniently under two headings, namely those originating in the lungs, the Hering-Breuer reflexes, and those originating in the carotid body, carotid sinus and aorta.

The Hering-Breuer reflexes are initiated at nerve endings in the alveolar ducts of the lung, nerve endings which are insensitive to physiological changes in the composition of the air of the lungs but highly sensitive to the effects of inflation which occurs with each inspiration. During a given inspiratory act, the frequency of impulses travelling up over vagal afferents increases until a critical rate is reached at which inspiration is inhibited and expiration begins. During expiration in quiet breathing the frequency of discharge of impulses is markedly reduced or abolished. Thus we are confronted with a reflex inhibition of the inspiratory neurones which increases with the progress of a given inspiration and decreases during expiration. The degree of vagal inhibition of inspiration then seems to be a function of the state of distention of the lungs, and functionally these fibers may be looked upon as a means of "notifying" the respiratory cells of the medulla of the degree of inflation of the lungs. Gesell[3] has shown that in the absence of the vagi the velocity of both inspiration and expiration is diminished.

We have pointed out that during expiration, in quiet breathing, the frequency of impulses travelling over vagal afferents from the lung is markedly reduced. However, if the lungs are abnormally collapsed as a result of introduction of air into the pleural space, or as a result of suction on the trachea, other vagal afferents are stimulated with the result that expirations are followed more promptly by inspirations. The result is an increase in respiratory rate due to stimulation

of excito-inspiratory, i.e., inspiratory, vagal receptors. There is some question as to whether these excito-inspiratory receptors are ever concerned with normal respiration, but it is assumed that under abnormal conditions such as atelectasis they may play a part in establishing the respiratory rhythm.

Respiratory reflexes originating in the carotid body,[2] carotid sinus[7,8] and aorta[6,8] comprise a relatively new chapter in the physiology of respiration, a chapter which made necessary a revision of some of the older concepts in which it had been assumed that changes in respiration produced by variations in carbon dioxide and oxygen tension were due entirely to central action on respiratory centers, and in which no attention was paid to impulses over afferent nerves from great vessels. These reflexes, it will be noted, originate in receptors located entirely outside the respiratory tract, in or closely associated with the blood vessels.

A rise of blood pressure in the carotid sinus and in the aorta of the experimental animal, or in either of these alone, results in either depression of respiration or apnea in expiration as well as in reflex cardiac slowing and loss of vasoconstrictor tonus. A fall of blood pressure, on the other hand, results in augmentation of respiration, rise in pulse rate and vasoconstriction. These responses are largely or completely abolished by denervation of the carotid sinuses and section of the depressor aortic nerves. In addition to these clear-cut effects of pressure on receptors in blood vessels, increasing numbers of experiments indicate that receptors which are sensitive to chemical changes in the blood also are located in the aorta, carotid sinuses and carotid bodies. Under experimental conditions, increased CO_2 tension, decreased O_2 tension or rise in acidity of arterial blood are capable of producing reflex augmentation of respiratory rhythm, while decreased CO_2 tension and fall in acidity tend to result in reflex depression of respiration. That these responses are mediated through the sinus and aortic nerves has been clearly demonstrated by Heymans and Heymans.[6]

The chemical and pressure changes necessary to elicit reflex effects on respiration, however, may well be greater than those occurring physiologically except in the case of oxygen lack. Anoxia, which has a direct depressing influence on the activity of cells in general, including those of the respiratory centers, interestingly enough has a stimulatory effect on chemoreceptors in the carotid body[2,10] which lies in close association with the carotid sinus, both structures being innervated by branches of the glossopharyngeal nerve. In severe anoxemia, with resultant depression of the respiratory cells of the medulla, respiration may cease entirely as a result of section of the nerves to the carotid body. Thus it appears that the carotid body receptors are responsible for a reflex augmentation of respiration in anoxemia, a condition which in the absence of this reflex would result in respiratory depression. The hyperpnea of experimental anoxemia then appears to be principally of carotid body reflex origin, while that of CO_2 excess and rise in acidity seems to be largely if not entirely associated with direct effects on the respiratory cells in the medulla.

The Chemical Control of Respiration

As already pointed out the role of anoxemia is concerned with a direct depressing influence of oxygen lack on the respiratory cells of the medulla, and an opposing excitatory effect upon chemoreceptors in the carotid body whose stimulation results in reflex augmentation of respiration. The respiratory neurones of the medulla, however, are extremely sensitive to variations in the CO_2 tension of the blood and somewhat less so to any other acids. In both cases the stimulatory effect concerns a rise in the hydrogen-ion concentration within the cells, CO_2 being more effective than other acids because it crosses cell membranes at a more rapid pace.

The importance of carbon dioxide in the control of respiration is properly emphasized by the results of experiments which have attempted to demonstrate the respiratory response to various concentrations of the gas. Such experiments in man[1] have shown that increases of 0.3 per cent in alveolar CO_2 are capable of increasing the volume of alveolar ventilation to as much as double its previous value.

As the percentage of carbon dioxide is gradually increased[4] alveolar ventilation, i.e., the difference between tidal air and dead space air increases at first to the extent that

alveolar carbon dioxide is prevented from rising. Above levels of about 4 per cent CO_2 in the inspired air, increases in alveolar ventilation are unable to keep pace and alveolar CO_2 begins to rise. Until this rise begins the increased alveolar ventilation is accomplished by increase in depth of respiration. After alveolar CO_2 begins to rise, respiratory rate increases, expiration is very active and dyspnea is evident.

It is clear then that small changes in alveolar CO_2 are capable of producing great changes in activity of the respiratory neurones of the medulla by direct influence on the hydrogen-ion content of these cells.

In sharp contrast to the influence of carbon dioxide is that of oxygen content of the inspired air. Administration of gas mixtures in which carbon dioxide remains relatively constant have shown that oxygen may be reduced from 80 per cent to 15 per cent without the development of significant hyperpnea.[4] Below about 12 per cent, hyperpnea may become fairly marked but not as great as in the case of CO_2 excess. Hyperpnea due to oxygen lack results in the washing out of CO_2 from the alveolar air, with consequent reduction of arterial blood CO_2 tension, or in other words the removal of the normal stimulus to the respiratory center. Thus hyperpnea of oxygen-lack is always counteracted by reduction of the usual stimulating effect of CO_2 with the result that this hyperpnea is not great. It seems quite obvious that oxygen lack is not a good stimulator of respiration, and in fact that it is not concerned with the regulation of respiration under usual conditions. It seems equally clear that the carbon dioxide of the blood is the most important chemical factor concerned with control of respiration. As CO_2 tension rises, the depth of respiration increases, even though the Hering-Breuer inspiratory-inhibitory reflex must be increasingly active, a result which again emphasizes the prepotent influence of CO_2.

The control of respiration under normal conditions then seems to involve the rhythmic discharge of impulses from respiratory neurones of the reticular formation of the medulla to the muscles of inspiration which in turn cause distention of the lungs to the point of stimulation of sensitive vagal endings concerned with the Hering-Breuer inspiratory-inhibitory effect. Inspiration is cut short and passive expiration follows smoothly. The frequency of discharge of the respiratory neurones and the number of fibers involved seems to be intimately associated with the hydrogen-ion concentration of these nerve cells, a concentration which depends largely on the CO_2 tension of arterial blood and the rate at which that blood flows through the medulla. Under usual conditions of quiet respiration the threshold for activation of the respiratory neurones concerned with expiration is not reached. However, if as in extreme exercise, CO_2 tension of blood rises to a higher level, this threshold is attained, active expiration occurs, and inspiratory Hering-Breuer reflexes may occur.

Reflexes originating from pressure changes in the aorta and carotid sinuses are thus far of only experimental interest. Anoxemia becomes a stimulus to respiration when oxygen of inspired air falls below about 15 per cent with resultant stimulation of chemoreceptors of the carotid body and reflex stimulation of the respiratory neurones. This reflex stimulation, however, results in a washing out of alveolar CO_2, reduction of arterial blood CO_2 tension and removal of the normal chemical stimulus of the respiratory centers. It serves to illustrate the possible effects of a great many other reflexes such as those concerned with pain in which the respiratory center may be reflexly driven to the point of lowering CO_2 tension below that point at which it serves as the chemical stimulus for respiration. In anesthesia this lowering of CO_2 tension may be accompanied by increase in the intake of the anesthetic gas which in turn lowers the sensitivity of the respiratory cells to CO_2. At this point, anesthetic removal of the pain impulses which were reflexly driving respiration, may leave the respiratory cells with a lower than normal sensitivity to CO_2 and supplied by a blood having lower than normal CO_2 tension. The result is respiratory arrest. CO_2 administration is, of course, of value in preventing this apnea. The widespread use of CO_2-O_2 mixtures in clinical medicine is a direct result of the recognition by Henderson[5] of the need for prevention of acapnia in just such conditions.

Respiratory depression or failure, its causes and the remedies for it are of more concern to the physician than are the details of the

normal regulation of respiration, and yet the former is most easily studied in the light of the latter. Of all the cells of the central nervous system those concerned with respiration and vasomotor control are most resistant to a variety of adverse conditions,[10] such as poisoning by cyanide or carbon monoxide, severe depression by narcotic drugs, increased intra-cranial pressure with resulting diminished cerebral circulation, and trauma or hemorrhage in the medulla. These adverse conditions have in common the effect of reducing oxidation in the respiratory neurones of the medulla either by depression of the process or by curtailing the supply of oxygen to them. In either case pulmonary ventilation is reduced and anoxemia further promoted. A vicious cycle is established in which the reduced activity of the respiratory neurones incident to their anoxia, promotes further anoxemia and anoxia through reduced lung ventilation. Under these conditions, CO_2 is no longer the potent stimulus that it is when the cells are normally reactive, and continuation of respiration may depend largely on reflexes originating in the chemoreceptors of the carotid body. It is in situations such as this that the need for respiratory stimulants is most acutely felt. And yet the objective which is quite obviously increased supply of oxygen to the respiratory neurones may well be reached more certainly by such an effective procedure as artificial respiration. The latter, it seems to me, has everything to offer in its favor. It acts to relieve an anoxemia which is responsible for the failure of the respiratory neurones to respond to normal stimuli. Respiratory stimulants, on the other hand, can at best initiate respiratory stimulation by attempting to artificially drive neurones which are already suffering from oxygen lack, to an increased activity requiring more oxygen which does not become available until after respiratory stimulation has taken place.

Of primary concern in the relief of respiratory depression, then, is the supply of oxygen to the respiratory neurones. The question of the importance of CO_2-O_2 mixtures in the relief of respiratory failure is not answered by assuming that the increase of CO_2 tension results in stimulation of the depressed respiratory neurones. In fact, it has been shown that frequently the administration of CO_2 alone may further depress a failing respiration, i.e., it may exert a narcotic effect on the respiratory neurones, when the latter are anoxic.[11] Administration of 5 per cent CO_2-O_2 mixtures are nonetheless valuable in preventing respiratory depression because they supply oxygen to the respiratory neurones and because they prevent CO_2 tension from falling to levels at which the respiratory neurones are deprived of their normal chemical stimulus. The treatment of severe respiratory depression must be designed both to improve the activity of the respiratory neurones and to furnish sufficient oxygen to other nervous structures and the heart in order to prevent permanent damage to these structures. Schmidt[10] has suggested that in combatting respiratory failure the most important problem is the supply of oxygen to the respiratory neurones. He suggests 5 requisites concerning attempts to relieve respiratory depression.

1) The supply of oxygen to the lungs must be insured by administration of O_2 or CO_2-O_2 mixtures, by inhalation if breathing is present, or by intratracheal insufflation or artificial respiration if there is apnea. A sufficiency of alveolar oxygen relieves the immediate emergency.

2) Steps must be taken to remove the cause of the depression, as for example in carbon monoxide poisoning, in which the removal of this gas is facilitated by breathing CO_2-O_2 mixtures.

3) The supply of blood to the brain must be maintained, i.e., a failing circulation demands steps to insure a sufficiently high arterial blood pressure to produce good circulation through the brain.

4) If intracranial pressure is high, steps should be taken to lower it as by the use of intravenous 50 per cent sucrose.

5) Body temperature should be kept up by artificial means if necessary.

Of all these procedures, the elevation of alveolar oxygen is the most important.

Summary

The control of respiration seems to be based on the following factors:

a) An intrinsic rhythm of the respiratory neurones of the medulla oblongata. This rhythm is dependent upon oxygen supply to the neurones involved. It is regulated by both reflex and chemical mechanisms.

b) The chemical regulation of respiration concerns the hydrogen ion content of the respiratory neurones which in turn is dependent upon the carbon dioxide tension of the blood and the rate of flow of blood through the medulla. Variations in blood oxygen tension under normal conditions are not thought to be concerned with direct regulating effects on the respiratory neurones. In severe anoxemia, however, the respiratory neurones are probably directly depressed as a result of anoxia.

c) The reflex control of respiration under normal conditions almost certainly is concerned principally with the Hering-Breuer vagal reflexes. The respiratory neurones may be driven reflexly, however, by impulses from the carotid bodies whose 9th nerve endings are stimulated by severe oxygen want. Similarly, respiration may be driven by impulses coming into the medulla over any afferent neurones as in pain, and over neurones from higher brain centers.

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Tuberculosis Case Finding*

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It is generally conceded that we are now in possession of sufficient knowledge, if properly and wisely applied, to control, if not to completely eradicate, the scourge of tuberculosis from amidst mankind as well as from among domesticated animals. Nor is there a lack of skilled personnel ready and eager to give battle to this foe of man and his lowly kin.

In the absence of a specific remedy, there are but three ways of combating a parasitic disease. One is to raise the resistance of the host to the invading organism; the second, is to prevent the entrance of the parasite into the body of the host; and thirdly, a combination of these two. In the earlier phases of the organized movement against tuberculosis the first was the method of choice or at least the one upon which chief reliance was placed. It was believed, and rightly so, that by improving the general health of the population, by means of good nutritution, oral and general hygiene, regular habits, fresh air, cleanliness, etc., tuberculosis could be resisted if not prevented. It was also believed that a slight infection with the tubercle bacillus confered a relative immunity to subsequent infection. Most authors still hold to that opinion. The anti-tuberculosis program then consisted mainly of health crusaders, nutrition classes, attention to teeth and tonsils, anti-spitting ordinances, general cleanliness, open air schools and preventoria. Then as now the child received the lion's share of attention. Meanwhile sanitation and the general standard of living have been greatly improved. Who is there

* Read before the Annual Meeting of the New Jersey Tuberculosis League, 1937, in New Brunswick, New Jersey.

to deny that these combined forces played an important and dominant role in the reduction of the tuberculosis mortality—from 200 per 100,000 in 1900 to 53 per 100,000 in 1935.[1] Other influences, to be sure, contributed their share toward achieving this result. The educational campaign, stressing all phases of anti-tuberculosis efforts in accordance with the best concepts prevailing at the time, attempts at early diagnosis, provisions for an ever increasing number of beds for the tuberculous in sanatoria and general hospitals (95,198 beds were available in 1935),[1] a corresponding increase in clinic and follow-up service, improved diagnostic procedures and techniques, particularly x-ray photography and fluoroscopy as well as the tuberculin test, collapse therapy by means of pneumothorax and the newer surgical methods, all of these played an important role, not only in reducing the mortality and morbidity but the incidence of infections as well. Earlier diagnosis, earlier hospitalization and in larger numbers of the sputum positive cases resulted in greatly lessened chances of spreading the infection. Thus was utilized the second possible method of combating the disease, by preventing the parasite from entering the body of the host.

It will undoubtedly always be necessary to use the combined methods of attack; i.e., raising and maintaining a high resistance of the host as well as the prevention of infection. It is on the latter, however, that we shall place our chief reliance in the future, more and more, for a complete control or eradication of the disease. Without the tubercle bacillus there can be no tuberculosis. We must also bear in mind, as Sir Newsholm pointed out many years ago, that tuberculosis is largely a quantitative disease. Given an infection with few bacilli the human host is capable of overcoming and localizing it with no resultant disease, leaving only a calcified nodule as an identifying mark and a relatively heightened immunity. A large dose of bacilli will produce mild or moderate disease, while a massive infection will produce severe destructive or even fatal disease. Dealing with tuberculosis in cattle, the principle of prevention of infection has been applied solely with great success in this country. Tuberculin testing of all cattle, eliminating or destroying all reactors, combined with pasturization of all milk has not only eliminated tuberculosis in cattle, but practically eradicated tuberculosis of bovine origin among humans, particularly that of gland, bone and joint tuberculosis.

While it is evident that we cannot apply the identical tactics to human beings, which are so efficient in controlling tuberculosis amongst cattle, yet the principle holds good. First and above all we must find the open case at the earliest possible moment. It is for this reason that case-finding technique and procedure assumes such great importance, all else is secondary and of little avail if this fails. Remembering that every case comes from some other case, to find that other case is the problem of the hour. Considering the knowledge and instrumentalities now in our possession, an effective and complete case-finding program, with a view toward controlling tuberculosis, would entail a periodic medical examination, including a roentgenogram or fluoroscopy of the chest, of every individual in the community. For economic as well as psychological reasons, such a procedure is obviously out of the question at present. It will take a long time to educate the public at large to the point where it will respond with the necessary funds and its whole-hearted cooperation.

Perforce then, we must restrict ourselves to certain groups in the community which past experience has taught us yield the largest number of active cases capable of spreading the infection. First of all, the contacts of known open cases. Secondly, those with symptoms and signs suspicious of tuberculosis usually referred to as suspects. Enlightened tuberculosis workers everywhere have for a long time concentrated on these two groups and will no doubt always continue to do so. The difficulty begins when we try to reach the apparently healthy section of the population, with no symptoms and no known contacts.

Study of statistical data reveals that the mortality of tuberculosis varies according to age groups. It is the leading cause of death between the ages of 15 and 45, the most productive period of human existence. Then again at the two extreme ends of life's span, in infancy, particularly in the first two years and again in the aged, at 75 and over. Sex differences are not sufficiently marked for

selective purposes, except in the femals between 15 and 25 where the mortality is 50 per cent higher than in the corresponding male group. Racial differences, however, are quite prominent, the general colored mortality being 3½ times that of the white. Economics and occupation play an extremely important role in the tuberculosis death rate, being seven times as great in the unskilled labor group as in the higher professions, 184.9 per 100,000 for the former as against 26.2 per 100,000 for the latter, according to Jessamine S. Whitney.[1] Then there are areas within the larger city limits, particularly where the population is greatest and housing conditions the poorest, where tuberculosis is most prevalent and its toll the heaviest.

It is pertinent to mention in this connection that the tuberculin testing and x-ray experience with school children of primary grade age shows that approximately 2500 children examined yield one active case of pulmonary tuberculosis; in children of high school age one in 500; college age group, one in 250. L. H. Ferguson quotes 1 per cent in High School ages, 15 to 25 years, and 6 to 1000 in the college group,[2] while in the adult, Brown of Albany[3] reports 4 per cent active pulmonary tuberculosis in parents of reactors and Edwards in New York City[4] found 20 per cent manifest lesions (15.26% active and 5% apparently arrested) in adults selected by means of a consulting clinic service.

In planning our case-finding program, we must consider not only what group will yield the largest number with open or active lesions, but also how to reach them. It is not at all easy to persuade a healthy individual to submit himself to a periodic health examination which must include x-ray film or fluoroscopy of the chest, entailing expenditure of time, effort and money, for preventive purposes only. We must, therefore, take advantage of whatever opportunities present themselves, whether these be legalistic, coersive, or psychologically persuasive, such as Board of Health ordinances affecting food handlers, domestics, barbers, beauticians, etc.; Public School rulings and rulings of higher institutions of learning, affecting school children, particularly of high school age; students in colleges and Universities, including their entire personnel (teachers, nurses, janitors, etc.). It is perfectly reasonable to expect, nay to demand, that patients admitted to hospitals of any character whatsoever receive the benefit of an x-ray examination of the chest unless the need for it is ruled out by a proper tuberculin test. It is imperative that the personnel of all hospitals, including nurses and internes, have a periodic tuberculin test and x-ray film of the chest. This holds true for inmates and employees of other public institutions, such as hospitals for the insane, epileptic colonies, penal and correctional institutions, etc. Other groups that might readily be reached by education or legal approach are public employees (municipal, county, state or federal) relief clients, colored groups, etc.

The problem must also be considered from the standpoint of administrative technique, of economics, of the relationship between physician and official board of health, as well as private health organizations.[4] A way must be found to correlate and coordinate these agencies to the end that they may function harmoniously and efficiently. In spite of the many phases and seeming complications it should not be impossible to work out a practical and satisfactory tuberculosis case-finding program based on the facts and suggestions elaborated in this discussion.

I have been requested to make special reference to case-finding in the colored. Since tuberculosis in the colored does not differ essentially from that in the white, either epidemiologically or pathologically, except for the likelihood of a more massive involvement and an acuter and shorter course in the former, the problem of case-finding in the colored must be carried out basically along the same lines as the white. It is only because of the fact that the tuberculosis mortality, morbidity and incidence of infection are so much higher in the negro, coupled with and largely dependent on his poorer economic status, and consequently a much lower standard of living, that we must give him special consideration.

Our educational efforts here must be greater, the search for the spreader of the infections more intense, the technique the simplest consistant with the best accepted standards, and above all easy of accomplishment. In order to gain the confidence of the colored for our program and the better to serve his interest we must give him every opportunity

to participate and encourage workers of his own race to shoulder the burden.

In Middlesex County, with the aid of our Tuberculosis League, we have put into operation a very simple program. We aim to give a fluoroscopic examination to every colored person that can be reached within the county at least once a year.

A full-time colored worker has been educating and organizing the colored people of this county with that aim in view during the past three months. She operates in a given community until all prospects are exhausted, then moves on to the next. The fluoroscope is transported to each community as required. The time is too short and the number of people examined too few to make any report on results. Our field is far from ideal, because our colored population is scattered over a large area and the total number is not over 6000. The project is frankly experimental but we hope in time to give a satisfactory accounting.

236 High Street.

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2 Ferguson, Lee H.: *Amer. Review of Tuberculosis*, October, 1937.
3 Brown, Wm. P.: *Archives of Pediatrics*, August, 1936.
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Correlation of Signs and Symptoms

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Rutland, Massachusetts

Another paper on the early diagnosis of tuberculosis might well appear as a further reiteration woven around a worn-out theme. The fact is, however, that the proportion of minimal cases admitted to the sanatoria remains substantially the same after many years of propaganda. It is also true that the diagnosis of a thoracic lesion is not always so simple as the printed page would indicate; yet there are symptoms and signs which point the way to a diagnosis and demand careful observation and study.

One explanation of late diagnoses is that the symptoms of tuberculosis occur not infrequently in other conditions and in similar combinations; secondly, that the patient and physician are both reluctant to face an unpleasant situation, as a result of which much time is frequently lost in an endeavor to reconcile the danger signs with other conditions rather than in an intensive effort to either prove or disprove the presence of tuberculosis.

It is no criticism of the doctor who is unable to make a diagnosis from a single examination. Observation and study over a considerable period of time are often necessary even for the specialist. Certainly, it is unfair to label a patient as tuberculous when possibly he is recovering from an attack of influenza. Granting that either disease may improve with a trip to the farm or a change of climate, it is far better to give the suspected individual the benefit of sufficient study with all the means available in most communities for a diagnosis, than for him and the doctor to be lulled into a false sense of security because of some apparent improvement following a hasty prescription of fresh air, milk and eggs. Despite laboratory facilities and mechanical appliances we are sometimes chagrined that we did not make better use of our eyes, ears, and whatever degree of judgment with which we were endowed.

Fifty years ago the family doctor heard the patient's story, listened with his ear through coat and shirt, and whatever he heard, if anything, was supplemented by his clinical sense or judgment. Doubtless there were many mistakes, but he made use of all the means at his disposal. Is that done now? The evidence available to the four senses: hearing, seeing, smelling and feeling, often overlooked or neglected, should be carefully weighed and not lightly cast aside because of a negative test or a slide rule formula. Symptoms and physical signs play an important part in the diagnosis of any disease, to say nothing of tuberculosis.

The evaluation of the physical signs: rales,

x-ray findings, blood changes, loss of weight, elevated temperature and pulse, and whatever other signs may be noted, must be determined by their relation to the symptoms which brought the patient to the doctor. An indifferent history may be a short cut to the diagnosis, but it may also lead to delay and a disastrous end result. A good history of symptoms is on the same level as a good physical examination and should never be relegated to second place. A diagnosis can often be made on the history alone with a small margin of error.

More mistakes are made through haste and failure to follow-up a lead than through ignorance alone. A diagnosis hinges on the history obtained—it is that which leads one to look for further evidence. More time and attention should be given to the story told by the patient; leading symptoms should be followed up to determine, if possible, their probable cause, duration, and their relation to other signs and symptoms. A history of contact with a fellow worker or some member of the family with a chronic cough may be a guiding point in the diagnosis.

The past history of the patient, particularly with regard to previous pulmonary infection, should be investigated. It may have an important bearing on the problem. Pleurisy occurring months or even years before is a bit of evidence which should be investigated. Pleurisy with effusion is usually of tuberculous origin and often may be linked with a series of so-called colds or exposure to a known case. Any case of pleurisy with effusion should be considered a potential case of tuberculosis and kept under observation pending a definite diagnosis.

The usual symptoms of tuberculosis are common knowledge. They are cough, fatigue, fever, accelerated pulse rate, hemorrhage, pains in the chest or shoulders, hoarseness, loss of weight and appetite, and digestive disturbances. Not one or any group of these symptoms is pathognomonic of the disease nor does the absence of any one, or group of them, eliminate the possibility of active disease.

The onset of tuberculosis may be most subtle, or it may develop with hurricane rapidity and violence. Definite lesions may exist for months, or even years, before symptoms have been noticed by the patient, as shown by follow-up examinations and x-rays of contact cases; or a hemorrhage may be the first symptom noted by the patient. Such a hemorrhage usually sends the patient to the doctor, and yet such tangible evidence of pathology has been ascribed to teeth, nasopharynx, sinuses, and vicarious menstruation. A so-called pneumonia which does not clear up as it should may be tuberculous as well as a series of so-called colds.

Frequently the subtlety of onset and the sense of well-being, as compared with many other diseases, obscure the presence of advanced disease before the danger is sensed either by the patient or by the physician. The symptoms are often vague and indefinite but a careful cross examination will indicate that something has happened which sent the patient to the doctor. For this reason, any one or any group of the common symptoms in a young adult should arouse a suspicion of tuberculosis and they demand a most careful investigation to determine the cause. Quite likely they may be due to conditions other than tuberculosis but the burden of proof rests with the doctor. More time should be devoted to history taking. It inspires confidence when a thorough investigation is made of the patient's personal problems, and with such a confidence established the physician oftentimes will get information otherwise kept in the background. Furthermore, he will get better cooperation in carrying out his recommendations or treatment. To get results there must be mutual confidence and cooperation between physician and patient, whether in the home or in the sanatorium, inasmuch as treatment does not end in a few days or weeks, but must be prolonged for months or even years.

Fatigue at the end of the day is physiological and for that reason may not receive serious attention, but fatigue in a young adult which does not disappear after rest and sleep, or the omission of night life merits an investigation. It is a common and early symptom of tuberculosis. Its relation to other symptoms and signs should be determined. In the absence of other symptoms, a blood examination, or an x-ray may indicate a probable cause. Unexplained fatigue with a history of a chronic cough in some member of the family, or a fellow worker, should not be overlooked. If accompanied by a loss of weight

or other symptoms it has still more significance. Hoarseness is often an early symptom and may easily be unnoticed. Either fatigue, cough or hemorrhage alone may be the first symptom noted by the patient. Frequently, a patient after admission to the sanatorium, will state that he has been treated for a cold for several months, or has consulted a physician for a run-down condition. He may not have had a physical examination of the chest or x-ray during this period until he changed doctors and discovered what he himself may have suspected.

A cough or recurrent colds continuing over a month is a common story and the possibility of tuberculosis should not be eliminated until one is certain of the underlying cause. A history of contact with a chronic cougher in the family may offer a clue. Acute respiratory infections should clear up in a few weeks, or at least show progressive improvement. A cough or chest cold lasting over a month should arouse suspicion and should be followed up by repeated examinations, including x-rays and sputum examinations; they should not be treated hit or miss with sedative cough mixtures which only mask the symptoms, and later patient and doctor far too often arrive at the sad truth after a minimal lesion has become caseated or excavated.

It is not enough to be satisfied with the mere statement that the patient has a cough, or a cold which does not clear up. A cough may signify little, or grave pathology from a nasopharyngeal irritation, to aortic aneurysm or tuberculosis. It should be investigated as to its character, whether hard or dry, or productive, and if productive, how much sputum, its character, color, consistency, and presence of blood streaks. Paradoxical as it may appear, patients will go to a physician for advice because of a cough, then fearing a diagnosis of tuberculosis will minimize its severity, productivity and duration. Frequently, its date of onset is placed with the first sputum noted, whereas a little cross examination may reveal that a "dry hack" may have been noticed for several months before the appearance of expectoration. With other symptoms, such a story has significance.

Blood streaked sputum should always be considered as coming from the lungs until definitely proved that its source is somewhere else in the respiratory or digestive tracts. Blood in the sputum usually means tuberculosis in a young adult. It is also a symptom of bronchiectasis, abscess, or cancer; conditions certainly as crippling, if not worse than tuberculosis. As a diagnostic sign, its relation to other symptoms and signs must be considered. Cancer of the lung, if taken in time can be cured, or at least relieved, but if treated for tuberculosis the results will inevitably be disastrous. In the case of abscess and of bronchiectasis, the therapeutic approach is again different. Symptoms oftentimes go unnoticed by the patient and frequently he can only approximate the date of appearance unless, perchance, to his surprise and terror, hemorrhage is the first to appear.

A loss of weight is a frequent symptom but may go unnoticed by the patient. It is usually accompanied by other symptoms, such as a poor appetite, digestive disturbances, or fatigue. Conversely, these patients may be surprisingly well nourished and maintain or gain weight for indefinite periods; in fact, they are quite likely to gain weight if rest and a diet are prescribed. While a gain of weight is desirable, one should not be misled by this sign alone. A loss of weight, as a rule, indicates active or progressive disease. A gain of weight is a favorable sign, but often deceives the patient and even the doctor as to the progress of the disease.

A loss of appetite and indigestion are symptoms common in many diseases and are significant in their relation to other evidence. A loss of appetite and digestive disturbances occurring during the course of the disease indicate a degree of activity either with or without demonstrable extension of lesions. During the early stages they usually indicate some functional disturbance which will respond to treatment directed to the underlying condition.

Fever is usually present some time during the 24 hours. Its absence at the time of an examination means little. The only way to be reasonably certain the patient is afebrile is to instruct him regarding the use of a clinical thermometer several times during the day and particularly during the evening. If this is done a transitory fever may be detected. What constitutes fever? Any persistent temperature of 99° Fahrenheit or over

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is fever even though it may be registered but once during the 24-hour interval. Such a fever should always be regarded with suspicion, either with or without symptoms. If there are symptoms, and there probably are or the patient would not have sought advice, it is an important bit of evidence however slight the temperature may be.

An increase of the pulse rate is usual but one should not be deceived by a rapid pulse rate due to nervousness or thyroid disease. The pulse rate taken at the beginning of an examination is quite likely to be accelerated, and at the end of the interview to be 10 to 15 beats less. A pulse rate 10 to 15 beats above normal for the age and sex, taken when the patient is at ease is significant.

The presence of tubercle bacilli in the sputum is the only positive evidence of the disease. Failure to find them in a smear in the presence of symptoms means little and should not defer a diagnosis which can be made from the history, physical signs, and x-ray with a very small percentage of error. A diagnosis should not be deferred in the presence of signs and symptoms pending the result of cultures or guinea pig inoculation unless the patient is put under treatment either in the home or a sanatorium. If this is done, it is quite possible that bacilli will never be found, but such negative evidence should not outweigh the clinical picture. We expect minimal cases, positive or negative for bacilli, to recover, but treatment should not be delayed pending scientific proof of the disease lest the case progress to cavity formation before such proof is obtained.

Next to a positive sputum, the x-ray comes the nearest to furnishing prima facie evidence of the disease, but in view of the varying technique in making a film, together with the varying experience of the interpreter, small lesions may be missed or the shadows interpreted as something else. No chest examination is complete without an x-ray. In many instances the picture may be characteristic; nevertheless, its interpretation should be linked with the history as in the case of any other sign of disease. The x-ray reveals evidence not demonstrable by the stethoscope; yet the stethoscope reveals rales and other signs not shown in a film. Both should be used together. A single film should not be relied upon to determine activity. A series of films may show progression or regression of a lesion. In either case the other signs and symptoms should be considered. A cottony shadow, particularly above or below the clavicle, without symptoms, should be regarded as probably tuberculous and treated as tuberculosis unless such a shadow is definitely proved to be something else. A history of contact or exposure to a known case of tuberculosis, or any of the symptoms, make the diagnosis almost a certainty. The absence of a known contact, however, means little or nothing. Given any combination or group of symptoms suggesting tuberculosis, even though cough is denied, an x-ray is indicated.

The physical signs of pulmonary tuberculosis may be as indefinite and illusive as the symptoms; they vary from a doubtful change of breath sounds to gross evidence of tissue destruction. About every auscultatory sound may be demonstrated in a single chest over a sufficient period of time. However, the first indefinite signs to appear, and the most difficult to detect, are the ones which point to beginning disease. While it is true there are combinations of rales, changes of resonance and respiratory sounds which usually mean tuberculosis, there is no single sign or combination that may not be heard in other conditions. Whatever the signs may be they should be correlated with the symptoms, x-ray and laboratory findings. Changes in the respiratory sounds, rales, and possibly a slight limitation of expansion on the affected side are among the earliest physical signs. They are often preceded by x-ray evidence and when found frequently indicate that the disease has passed the minimal stage. Directing the patient to breath out, cough, and then to breathe in will often reveal rales which are not audible during ordinary respiration. The location of rales is of more importance than whether they are fine or coarse in character. Rales in the upper third of the chest, particularly those above and below the clavicle and at the scapular spine, should be considered due to a tuberculous lesion until proved otherwise. They do not indicate the degree of activity or necessarily the extent of the invasion; these facts must be determined by a study of the symptoms and x-ray. Rales above or below the clavicles in com-

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bination with any of the usual symptoms demand intensive study and immediate treatment, which may be carried out, in some measure at least, in the home. Time should not be lost pending the demonstration of the tubercle bacillus, which may require a gastric lavage and culture of the gastric contents.

Distressing as it may be, the patient should be told as early as possible that he has tuberculosis or even suspected tuberculosis. To the patient who knows the possibilities of the disease the information may come as a shock, but it is in his interest and a kindness to inform him at the earliest moment rather than to gamble on the chance that the disease may be arrested without his knowledge. It is a greater shock to be later told the truth, possibly by another physician, than to be told in the beginning. In either case confidence is shaken and resentment lingers. Cooperation of the patient is absolutely necessary if satisfactory treatment is to be carried out either in the home or in the sanatorium, and intelligent and willing cooperation cannot be maintained unless the patient knows the problem and some of the answers.

It is not necessary that all the leading or usual symptoms be present in order to make a diagnosis. One may often forsee in some measure the picture of a jigsaw puzzle before the pieces are all fitted together. By the same token, a group of symptoms, two or more, may point the way to a tentative diagnosis, or at least to the necessity for prolonged observation and study.

A diagnosis should not end merely with the proof of existing tuberculosis. It must be carried a step further if rational treatment is to be carried out. The duration and extent of the disease, the degree of activity, the age of the patient, and his economic status are all factors requiring careful study, and frequently cannot be determined in the home. It is evident that the treatment of an early and active lesion in a young adult differs from that of an old fibroid case discovered by chance in some clinic, or in a checkup of the patient's family. The first may require all that we have to offer, while the latter may need little or nothing save some guidance in the mode of living.

The treatment of tuberculosis is no longer a one man job. It calls for the combined judgment of the internist, surgeon, and other specialists. Suffice it to say, that the first step is the education of the patient as to why a regulated life is the background for any procedure that may be instituted; that it is how one lives rather than where if lasting results are to be achieved. For the vast majority this first step, the education of the patient, should begin in the sanatorium as a preparation to carry on after the return to the home and community.

Rutland State Sanatorium.

Organization News

OFFICERS ELECTED

The following officers were elected at the annual meeting of the American College of Chest Physicians held at Cleveland, Ohio, May 31-June 2, 1941:

President Elect:
Dr. J. Winthrop Peabody, Washington, D. C.

First Vice-President:
Dr. J. Arthur Myers, Minneapolis, Minnesota.

Second Vice-President:
Dr. Grover C. Bellinger, Salem, Oregon.

Secretary-Treasurer:
*Dr. Paul H. Holinger, Chicago, Illinois.

* Reelected.

Regents

District
No. 1. *Dr. Moses J. Stone, Boston, Mass.
No. 4. *Dr. Paul H. Ringer, Asheville, N C.
No. 7. Dr. H. I. Spector, St. Louis, Missouri.
No. 12. Dr. Chas. H. Kibler, Tucson, Arizona.
No. 13. Dr. Harry C. Warren, San Francisco, Cal.
No. 14. Dr. Frederick Slyfield, Seattle, Wash.
No. 15. *Dr. Antonio Navarrete, Havana, Cuba.

Governors

Arizona—Dr. Hilton J. McKeown, Phoenix.
Arkansas—Dr. David H. Shipp, Little Rock.
Dist. of Columbia—Dr. William D. Tewksbury, Washington.
Florida—Dr. M. Jay Flipse, Miami.
Colorado—Dr. Arnold Minnig, Denver.
Idaho—*Dr. Orval A. Swindell, Boise.
Kansas—Dr. Arthur L. Ashmore, Wichita.
Maine—*Dr. Edward A. Greco, Portland.
Maryland—Dr. Victor F. Cullen, State Sanatorium.
Michigan—Dr. William A. Hudson, Detroit.
Missouri—Dr. Elmer E. Glenn, Springfield.
N. Carolina—*Dr. Karl Schaffle, Asheville.
Ohio—*Dr. Louis Mark, Columbus.
S. Dakota—*Dr. Lester D. Riggs, Fort Thompson.
Texas—Dr. Chas. J. Koerth, San Antonio.
Virginia—*Dr. Dean B. Cole, Richmond.
Washington—Dr. John E. Nelson, Seattle.
W. Virginia—Dr. George F. Evans, Clarksburg.

* Reelected.

Canal Zone—*Dr. Mack M. Green, Panama.
Puerto Rico—*Dr. Jacob Smith, Rio Piedras.
Philippine Islands—*Dr. Miguel Canizares, Manila.
Australia—*Dr. John H. Blackburn, Queensland.
Canada—Dr. William E. Ogden, Toronto.
China—*Dr. Shu-Fan Li, Hong Kong.
Cuba—*Dr. Octavio Rivero, Havana.
Ecuador—*Dr. Juan Tanca Marengo, Guayaquil.
India—*Dr. Jaharlal Ghosh, Calcutta.
Mexico—*Dr. Donato G. Alarcon, Mexico City.
Norway—*Dr. Carl B. Semb, Oslo.
S. Africa—*Dr. David Pieter Marais, Cape town.

Dr. Benjamin Goldberg, Chicago, Illinois, succeeds Dr. John H. Peck, Oakdale, Iowa, as the President of the American College of Chest Physicians.

STATE CHAPTERS

New York State Chapter

At a meeting of the New York State Chapter held at Cleveland, June 2nd, in connection with the annual meeting of the American College of Chest Physicians, the following officers were elected: Dr. Nelson W. Strohm, Buffalo, President; Dr. Jas. S. Edlin, New York City, Vice-President; and Dr. Arthur Q. Penta, Schenectady, Secretary-Treasurer. The meeting was addressed by Dr. Benjamin Goldberg, President of the American College of Chest Physicians.

New Jersey Chapter

The annual meeting of the New Jersey Chapter of the College was held at the Hadden-Hall, Atlantic City, New Jersey, on May 21st, in connection with the annual meeting of the New Jersey State Medical Society. The following officers were elected: Dr. Joseph R. Morrow, Ridgewood, President; Dr. Clyde M. Fish, Pleasantville, Vice-President; and Dr. Charles I. Silk, Perth Amboy, was reelected as Secretary-Treasurer.

Dr. Martin H. Collier, Grenloch, President of the Chapter, presided at the meeting and he introduced Dr. Frank Walton Burge, Philadelphia, Chairman of the Board of Regents of the College, who gave a talk on "Pneumoperitoneum" illustrated by x-ray films.

Pennsylvania Chapter

The Pennsylvania Chapter of the College was organized at Cleveland on June 2nd in conjunction with the annual meeting of the American College of Chest Physicians. The following officers were elected protem: Dr. R. S. Anderson, Erie, President; Dr. Harry J. Treshler, Cresson, Vice-President; and Dr. Edward Lebovitz, Secretary-Treasurer. The Pennsylvania Chapter will meet at Pittsburgh in the Fall of the year and permanent officers for the year will come up for election. The meeting was addressed by Dr. John H. Peck, Oakdale, Iowa, Past President of the College.

Ohio Chapter

The Ohio Chapter of the American College of Chest Physicians was organized at the Hollenden Hotel, Cleveland, June 2nd. The following officers were elected: Dr. Louis Mark, Columbus, President; Dr. John H. Skavlem, Cincinnati, Vice-President; and Dr. Joseph B. Stocklen, Secretary-Treasurer. Dr. J. Winthrop Peabody, Washington, D. C., President Elect of the College, addressed the meeting.

REPORT OF THE RESOLUTIONS COMMITTEE

RESOLVED: that the American College of Chest Physicians does hereby express to Dr. Joseph C. Placak and to the members of his committee, and in particular to his Section Chairmen: Dr. Raymond C. McKay, Dr. Samuel O. Freedlander, Dr. Warren C. Breidenbach, and Dr. John H. Skavlem; and to Dr. Paul W. Gebauer and his committee and to Dr. Louis Mark and his highly successful entertainment committee; and to Dr. Benjamin Goldberg and his committee who arranged an excellent scientific program; and to all others who took part or assisted in the arrangements for this seventh annual meeting of the American College of Chest Physicians; its very greatful thanks and appreciation for their work in bringing about a highly successful annual convention.

John H. Peck, Oakdale, Iowa,
Victor S. Randolph, Phoenix, Arizona,
Harry C. Warren, San Francisco, California.

DISTRICT MEDICAL SOCIETY MEETS AT McCONNELSVILLE

The Eighth District Ohio State Medical Society held its annual meeting at the Rocky Glen Sanatorium, McConnelsville, Ohio, on June 19th.

Dr. Leon Schiff, Cincinnati, Ohio, read a paper on "Hematemesis and Melena" (Newer Concepts) and Dr. Claude S. Beck, Cleveland, Ohio, read a paper on "Heart Diseases Treated by Surgical Methods." Dr. A. A. Tombaugh, McConnelsville, Ohio, President of the Eighth District Medical Society presided.

Dr. Louis Mark, Medical Director of the Rocky Glen Sanatorium was host to the Eighth District Medical Society. Dr. Mark was recently elected President of the Ohio State Chapter of the American College of Chest Physicians and he is the Governor of the College for the State of Ohio.